Food-borne Illness: Bacterial

Gastrointestinal disturbances resulting from the ingestion of food can have a variety of causes, e.g., overeating; allergies; nutritional deficiencies; actual poisoning by chemicals, toxic plants, or animals; toxins produced by bacteria; infestation by animal parasites; and infection by microorganisms. These illnesses often are grouped together because they have rather similar symptoms at times and sometimes are mistaken for each other. This chapter will discuss food-borne diseases of which the etiologic agents are bacteria.

FOOD-BORNE DISEASES

Ordinarily, the term "food poisoning," as applied to diseases caused by microorganisms, is used very loosely to include both illnesses caused by the ingestion of toxins elaborated by the organisms and those resulting from infection of the host through the intestinal tract. A further classification of food-borne diseases is shown in Figure 24.1. Here, all food-borne diseases are subdivided into poisonings and infections. Food poisonings can be the result of either chemical poisoning or the ingestion of a toxicant (intoxication). The toxicant might be found naturally in certain plants or animals (see Chapter 25) or be a toxic metabolic product excreted by a microorganism. A **bacterial food intoxication** therefore refers to food-borne illnesses caused by the presence of a bacterial toxin formed in the food. **A bacterial food infection** refers to food-borne illnesses caused by the entrance of bacteria into the body through ingestion of contaminated foods and the reaction of the body to their presence or to their metabolites.

According to this classification, there are two chief kinds of food intoxications caused by bacteria (see Figure 24.2): (1) botulism, caused by the presence in food of toxin produced by *Clostridium botulinum*, and (2) staphylococcal intoxication, caused by a toxin in the food from *Staphylococcus aureus*.

Food infections listed in Figure 24.2 can be divided into two types: (1) those in which the food does not ordinarily support growth of the pathogens but merely carries them, i.e., pathogens such as those causing tuberculosis, diphtheria, the dysenteries, typhoid fever, brucellosis, cholera, infectious hepatitis, Q fever, etc., and (2) those in which the food can serve as a culture medium for growth of the pathogens to numbers that will increase the likelihood of infection of the consumer of the food; these include *Salmonella* spp., *Vibrio parahaemolyticus*, and enteropathogenic *Escherichia coli*. Outbreaks of food infections of the second type are likely to be more explosive than outbreaks caused by other intestinal pathogens.

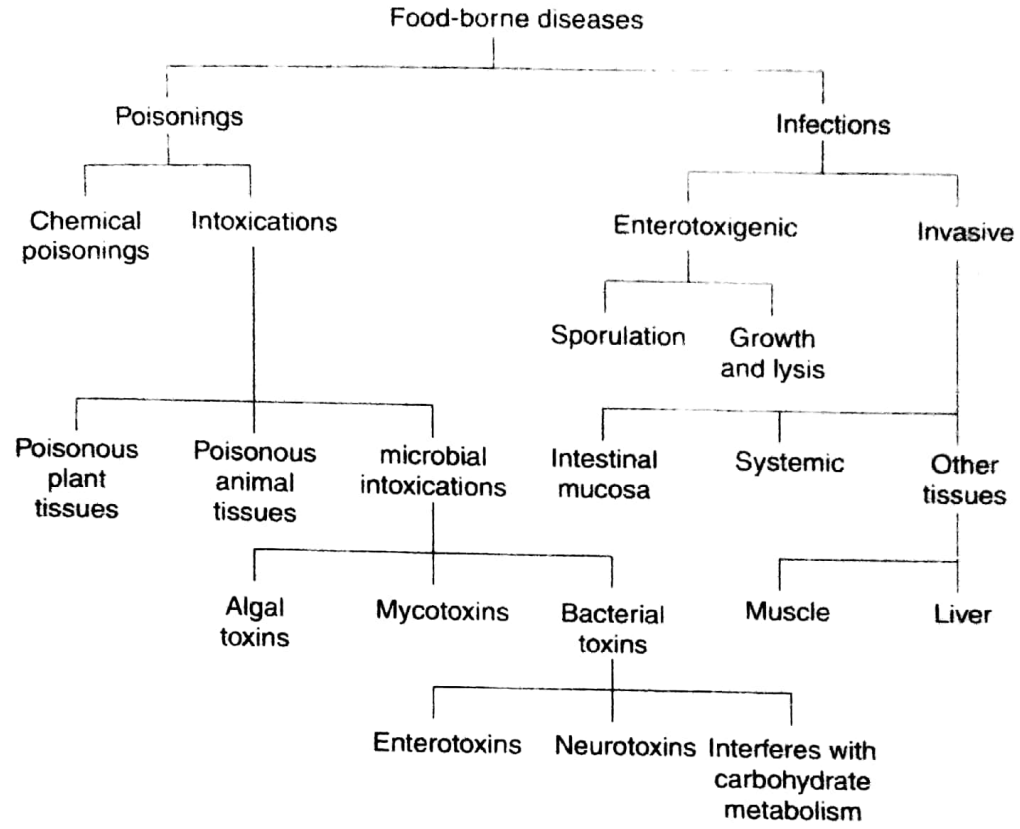

Fig. 24.1 *A classification of Food-borne Diseases. (Adapted from Bryan, F. L. 1976. Diseases Transmitted by foods. DHEW Pub. No. (CDC) 76–8237, Center for Disease Control, Atlanta, Ga.)*

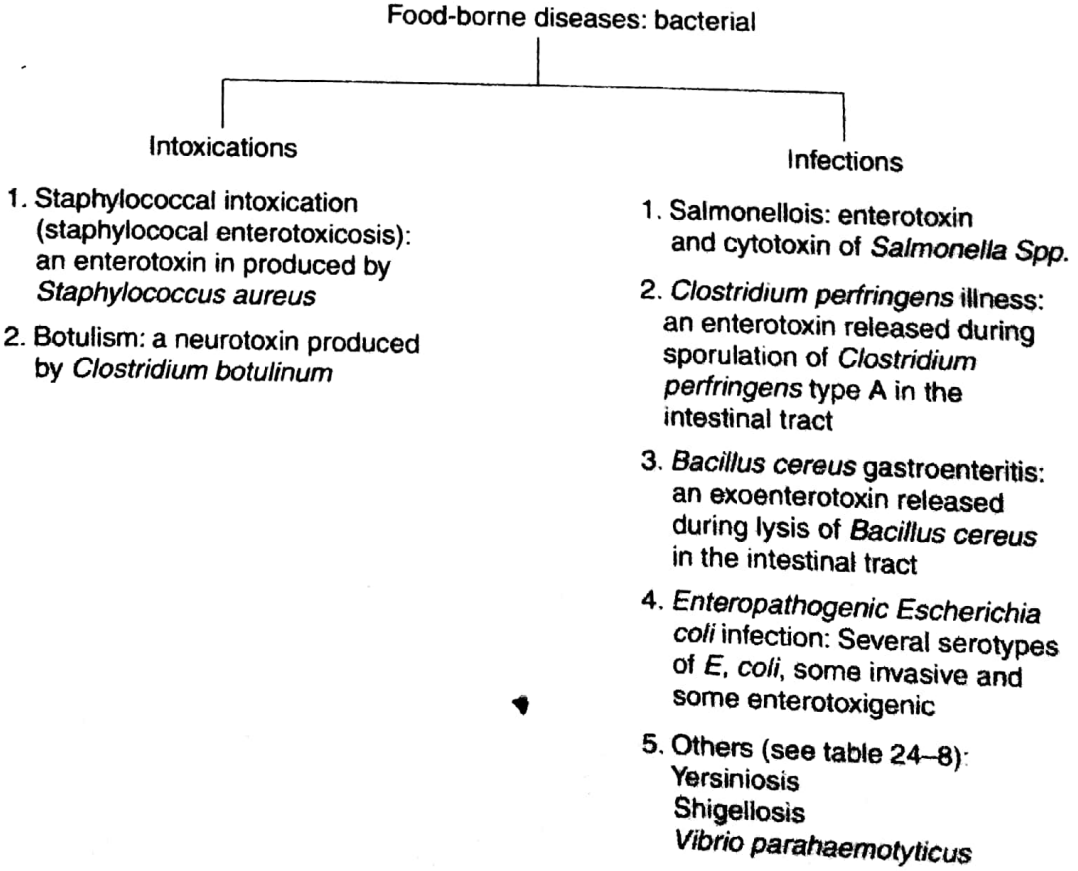

Fig. 24.2 *Examples of Bacteria Responsible for Food-borne Intoxications and Infections.*

Some workers suggest that *Clostridium perfringens* food-borne illness and *Bacillus cereus* gastroenteritis should be listed as food intoxications rather than food infections since toxin *might be* released by *B. cereus* as a result of autolysis of the cells in the food or by *C. perfringens* during sporulation in the food. If the toxins were released in the food, they would be better classified as food intoxications. However, as is discussed in the following sections, a large number of viable cells must be consumed in both cases, which implies the release of toxin in vivo rather than in the food. The controversial status of these two food-borne illnesses is recognized. Additionally, the whole categorization into food infections and food intoxications is not as straightforward as it once was. *S. aureus* produces an enterotoxin in the food and is frequently referred to as the classical food intoxication. However, several gram-positive bacteria, including *S. aureus, C. perfringens,* and *B. cereus,* can colonize the intestinal mucosa and be responsible for chronic diarrhea. Also, the pathogenicity of *Salmonella* is thought to be due to an enterotoxin and perhaps a cytotoxin. Therefore, these terms should be used cautiously.

Table 24.1 shows the number of outbreaks and cases during a 4-year period in the United States from several bacterial agents. These figures probably represent only a fraction of the outbreaks and cases that really occurred during each period. Since many people do not consult a physician about some diarrheal illnesses, health departments and normal surveillance channels would not be aware of the incident, and therefore it would not be reported and tabulated. The majority of outbreaks and cases were attributed to staphylococcal intox- ication, salmonellosis, and *Clostridium perfringens* gastroenteritis.

Despite the efforts of many agencies, industries, and people, outbreaks continue to occur. Table 24.2 summarizes data regarding the location or site of food abuse or mishandling that resulted in reported food-borne outbreaks from 1968 to 1976.

Table 24.1 *Some Food-borne Illnesses Reported in the United States, 1978–1982*

Disease	1978	1979	1980	1981	1982
Staph, lococcal intoxication	–	2,391(34)	944(27)	2,934(44)	669(28)
Clostridium perfringens gastroenteritis	617(9)*	1,110(2)	1,463(25)	1,162(28)	1,189(22)
Salmonellosis	1,921(45)	2,794(44)	2,381(39)	2,456(66)	2,056(55)
Botulism	58(12)	9(7)	18(14)	22(11)	30(21)
Bacillus cereus gastroenteritis	248(6)	–	187(9)	74(8)	200(8)
Shigellosis	159(4)	356(7)	1,184(11)	351(9)	116(4)
Vibrio parahaemolyticus gastroenteritis	86(2)	14(2)	12(4)	13(2)	39(3)
Escherichia coli gastroenteritis	35(1)	–	500(1)	–	47(2)

* Number of cases followed by number of outbreaks in parentheses

Source: Center for Disease Control, U.S. Department of Health, Education and Welfare. Foodborne Outbreak Annual Summaries.

Table 24.2 *Places Where Foods were Mishandled 1968–1976*

Place	*Number*	*Percent*
Food service establishments	1,283	39
Homes	504	15
Food processing plants	163	5
Unknown or unspecified	1,354	41
Total	3,304	100

Source: Center for Disease Control, U.S. Department of Health, Education and Welfare, Foodborne Outbreak Annual Summaries.

Food processing plants were responsible for 5 percent of the outbreaks, but for the majority of outbreaks the site of mishandling could not be specified. Food service establishments were responsible for over one-third of the food-borne outbreaks in 1968–1976.

❑ Botulism

Botulism is a disease caused by the ingestion of food containing the neurotoxin produced by *Clostridium botulinum.*

The Organism This rod-shaped soil bacterium is saprophytic, spore forming, gas forming, and anaerobic. Seven types are distinguished on the basis of the serological specificity of their toxins; the predominant (or only) toxin from these types is designated by the same capital letter.

Type A is the one commonly causing human botulism in the western part of the United States. It is more toxic than type B.

Type B is found more often than type A in most soils of the world and is less toxic to human beings.

Type C causes botulism of fowls, cattle, mink, and other animals but not of human beings so far as is known.

Type D is associated with forage poisoning of cattle in the Union of South Africa.

Type E, which is toxic for humans, has been obtained chiefly from fish and fish products.

Type F, which except for its toxin is similar to types A and B, has been isolated in Denmark and produces human botulism.

Type G has been isolated from the soil in Argentina but has not been implicated in human botulism.

Not all types produce a single toxin. For example, some type C strains produce predominately C_1 toxin with smaller amounts of D and C_2, or only C_2. Type D strains produce predominately D toxin with lesser amounts of C_1 and C_2 toxins.

Type A strains and most cultures of type B are proteolytic and are putrefactive enough to give an obnoxious odor to proteinaceous foods, but some strains of type B and those of type E are not. Even the first two types fail to give marked indications of putrefaction in low-protein foods such as string beans and corn, although they

produce toxin. The organism ferments carbohydrates with gas production, but sometimes this is not evident either.

Separation of the various strains of *C. botulinum* solely on the basis of toxin types results in very heterogeneous groups. For this reason, *C. botulinum* strains frequently are divided into three general groups, based on cultural and physiological characters, as follows:

Group I includes all type A strains (proteolytic) and the proteolytic strains of B and F.

Group II includes all type E strains (nonproteolytic) and the nonproteolytic strains of B and F.

Group III includes types C and D; they are nonproteolytic and share a common metabolic pattern.

Growth and Toxin Production Toxin production by *C. botulinum* depends on the ability of the cells to grow in a food and to autolyze there, for the types *A*, *B*, *E*, and *F* toxins apparently are synthesized as large, comparatively inactive proteins which become fully toxic after some hydrolysis. Therefore, the factors that influence spore germination, growth, and hence toxin production are of special interest. These factors include the composition of the food or medium, especially its nutritive properties (e.g., glucose or maltose is known to be essential for toxin production), moisture content, pH, O-R potential, and salt content, and the temperature and time of storage of the food. It is the combination of these factors that determines whether growth can take place and the rate and extent of that growth. Thus the nutritive properties of the food are likely to determine the minimal pH or temperature and the maximal concentration of sodium chloride for growth and toxin production. Results will differ with the serological type of organism and the particular strain.

Although foods are known to differ as culture media for *C. botulinum*, much of the evidence is empirical. Most of the studies have been on toxin production in various foods. Meats, fish, and low-or medium-acid canned foods have been shown to support toxin production and to differ in the potency of the toxin formed. Even good culture media may differ in the relative potency of the toxin formed in them. It has been reported, for instance, that media containing milk or casein, glucose or maltose, and corn-steep liquor yield more potent type A toxin than other media and that potencies of toxin from the following canned foods are, in descending order, corn>peas>string beans>spinach. Dissolved tin from cans has been shown to inhibit growth and toxin production in canned vegetables. Experiments on dehydrated meat have shown that toxin was produced more slowly when the moisture content was 40 percent than when it was 60 percent and that reduction to 30 percent prevented toxin production.

The concentrations of sodium chloride necessary to prevent growth and toxin production in foods depend on the composition of the food and the temperature. The presence of sodium nitrate in sausage or of disodium phosphate in cheese spread reduces the level of sodium chloride necessary to prevent toxin production. More salt is needed at a higher temperature, such as 37 C, than at a lower one, say, 15 C. Under favorable conditions for growth 8 percent or more of salt is needed to inhibit *C. botulinum*.

A pH near neutrality favors *C. botulinum*. The minimal pH at which growth and toxin production will take place depends on the kind of food and the temperature.

A pH of 4.5 or lower will prevent toxin production in most foods, but the lowest pH for spore germination is considerably higher. Minimal pH values reported are 4.87 for vegetative cells and 5.01 for spore germination in a veal infusion broth, 4.8 to 5.0 in bread, and 4.8 in pineapple-rice pudding. A maximal pH of 8.89 was found for vegetative growth. *C. botulinum* has been found growing and producing toxin, however, in foods that normally are too acid for it, when other microorganisms also were growing in the food and presumably raising the pH locally or generally.

There have been outbreaks of botulism from inadequately heat-processed canned high-acid foods (pH less than 4.5) including tomatoes, tomato juice, and blackberries. Possible explanations for these outbreaks have included (1) growth of other organisms which could raise the pH of a food so that *C. botulinum* could grow, (2) growth of *C. botulinum* followed by growth of other organisms which lowered the pH of a food that originally had a pH higher than 4.5, and (3) variation or stratification of the pH in an acidulated product to permit growth of *C. botulinum*. Tanaka (1982) has reported toxin production in media at a pH lower than 4.6.

Temperature is an important factor in determining whether toxin production will take place and what the rate of production will be. Vegetative growth will take place at a lower temperature than the minimum for spore germination. Different strains of *C. botulinum* types A and B vary in their temperature requirements. A few strains have been reported able to grow at 10 to 11 C, but about 15 C has been claimed to be the lowest temperature for germination of spores. The maximal temperature for growth is about 48 C for these types and about 45 C for type E. Type E organisms produce gas and toxin within 31 to 45 days at temperatures as low as 3.3 C.

The optimal temperature for toxin production and growth of the proteolytic strains is about 35 C, while 26 to 28 C is usually given for the optimal temperature for the nonproteolytic strains. Obviously, the slower the rate of toxin production, the longer it will take to obtain appreciable amounts. Interesting work (Lund et al., 1985) has begun to describe the interactions between pH, O-R, and temperature in regard to growth and toxin production.

The Toxin The toxin of *C. botulinum*, a protein that has been purified and crystallized, is so powerful that only a tiny amount is sufficient to cause death. It is absorbed mostly in the small intestine and paralyzes the involuntary muscles of the body. An important characteristic is its comparative thermolability. The heat treatment necessary to destroy it depends on the type of organism producing the toxin and the medium in which it is heated. In the laboratory, heat treatments of 5 to 6 min at 80 C will inactivate type A toxin, while 15 min at 90 C will inactivate type B toxin. This should not imply that the thorough cooking of a highly suspect food would be a worthwhile risk. As previously stated, the growth of *C. botulinum* in some foods results in such a foul, rancid odor that they would be rejected. Meats and proteinaceous, low-acid vegetables develop an especially obnoxious odor. More acid foods, however, and those low in proteins may become just as toxic without much evidence of putrefaction. Moreover, the nonproteolytic strains of *C. botulinum* give less evidence of spoilage than do the proteolytic ones. Also, gas production is not always evident and therefore is not a reliable indication of spoilage by this organism. Certainly it is advisable to reject all foods, raw or canned, that give evidence of spoilage and to reject canned foods that exhibit any pressure in the container.

The toxin can be destroyed in cheese by 7.3 Mrad of gamma rays and in broth by 4.9 Mrad. The toxin has been known to persist in foods for long periods, especially when storage has been at low temperatures. It is unstable at pH values above 6.8.

The nonproteolytic strains produce toxins which are not fully activated, and the addition of trypsin will result in a greater maximum potential for toxicity.

As has been indicated, the seven toxins (A to G) are antigenic, causing the production of antitoxin specific for a given toxin type injected. Toxoids have been prepared for some of the types for the active immunization of researchers who might be exposed to accidental poisoning by the toxin of botulism.

Toxicity and Bacteriophages It is not uncommon to isolate nontoxigenic colonies from a known toxigenic strain of *C. botulinum*. Recent studies on the relationship between toxigenicity (ability to produce the toxin) and temperate bacteriophages (intracellular, integrated phage nucleic acid) have suggested that the bacterial genome may not be responsible for the production of the toxin but is coded for by the genome of an incorporated temperate bacteriophage. This would explain the occasional loss of toxigenicity by some strains. Experimentally, types C and D can be "cured" of their temperate bacteriophage and become nontoxigenic. Types A, B, and F have been "cured" of bacteriophages also but have remained toxigenic. Apparently, there also exist bacteriophages for *C. botulinum* which impart toxigenesis. Also, one strain may harbor more than one temperate bacteriophage (multiple infection), and this may account for the fact that some strains produce more than one type of toxin, as mentioned previously.

Heat Resistance of Spores Compared with the spores of most other *Clostridium* species, those of some of the putrefactive anaerobes, including *C. botulinum*, have a comparatively high resistance to heat. The heat treatment necessary to destroy all the spores in a food depends on the kind of food, the type and strain of *C. botulinum*, the medium in which the spores were formed, the temperature at which they were produced, the age of the spores, and the numbers of spores present. The reader should refer to Chapter 6 for a discussion of the factors that influence the heat resistance of spores. Esty and Meyer (1922) have recommended the following heat treatments to destroy all spores of *C. botulinum* in a food:

Temperature, C	*Time, min*
100	360
105	120
110	36
115	12
120	4

In general, spores of organisms of types C, D, and E are less heat-resistant than those of types A and B, type E spores being inactivated in 15 min at 80 C.

Types A and B spores have D_{121C} values of 0.21 min while the D_{100C} value for type E has been reported to be 0.003 to 0.017 min. A comparison of type C spores has shown that marine strains are more heat-resistant than are terrestrial strains (D_{104C} of 0.4 to 0.9 min for the former and D_{104C} of 0.02 to 0.08 for the latter).

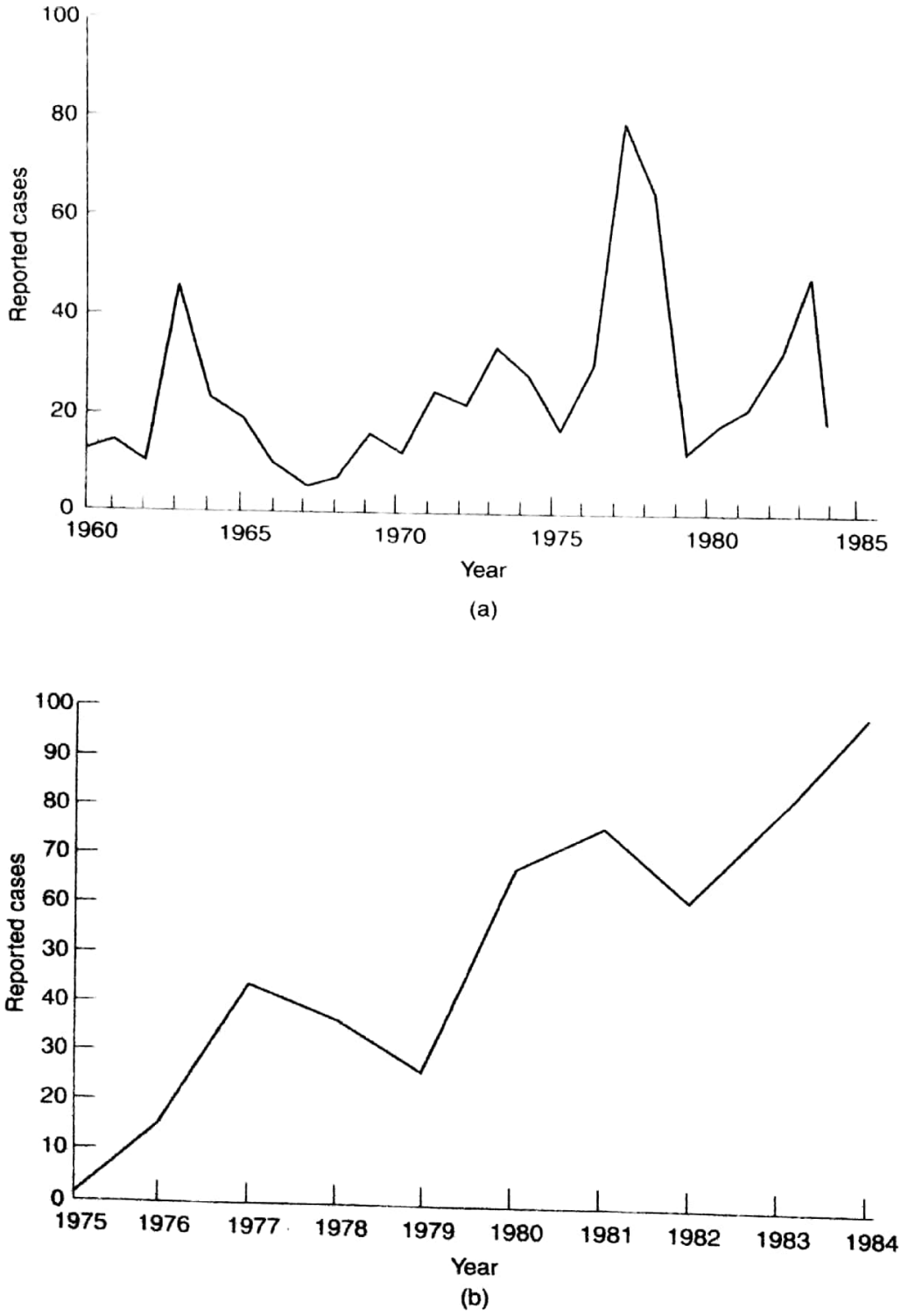

Fig. 24.3 *Reported cases of botulism per year in the United States. (A) Food-borne. Sixteen outbreaks (19 cases) of food-borne botulism were reported for 1984. Two of these outbreaks involved four individuals and were associated with eating fermented foods. Type E toxin was implicated. Type A toxin was associated with thirteen of the remaining cases. Type B toxin was associated with one case, and for the other case a toxin type was not determined. (B) Infant botulism. Of the ninety-nine infant botulism cases reported in 1984, slightly more than half (fifty-six) were in females. The age range for all petients was 3 to 37 weeks. Type A toxin was found in forty-two (42 percent) of the cases, type B toxin in fifty-six (57 percent), and both type A and type B toxins in one. (Center for Disease Control, Annual Summaries for 1984, 1986.)*

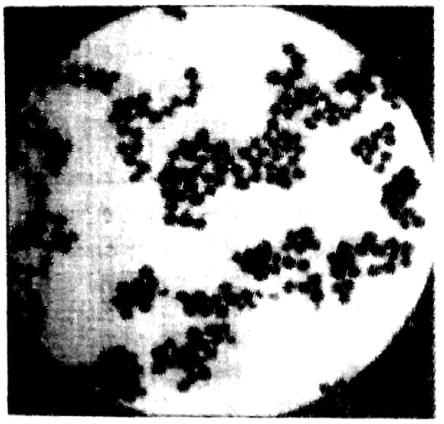

Fig. 24.4 *Photomicrograph of Staphylococcus aureus. (From J. Nowack.)*

The range of conditions permitting growth of the staphylococcus, and hence toxin production, varies with the food involved. In general, the better medium the food is for the coccus, the wider the range of temperature, pH, or a_w over which growth can take place. The temperature range for growth and toxin production is about 4 to 46 C, depending on the food. Mean generation times in several foods are shown in Figure 24.5. *S. aureus* grows most rapidly between 20 and 45 C; however, growth is four times more rapid at 45 C than it is at 20 C (Figure 24.5). The minimal temperature in custard, condensed milk, and chicken a la king is about 6.7 to 7.8 C, but it is about 10 C in beef-heart infusion and is higher in ham salad. Of course, growth at these minimal temperatures is very slow, and the time to attain numbers sufficient for production of detectable levels of toxin is longer than the storage period for most refrigerated foods. The minimal pH for growth is lower under aerobic conditions than under anaerobic; e.g., in meat the minimal pH is 4.8 in aerobic conditions and about 5.5 in anaerobic, while the maximal pH for growth is about pH 8.0. The minimal a_w is about 0.86 under aerobic conditions and about 0.90 under anaerobic conditions. A sublethal heat treatment decreases tolerance to salt.

Other kinds of food bacteria, competing with the staphylococcus, may repress its growth enough to delay or prevent production of toxin, or the spoilage bacteria may make the food inedible before it is dangerous. The effectiveness of the repression varies with the kinds and numbers of competing organisms, the type of food, temperature, and time. Ordinarily staphylococci enter foods in low numbers and usually are outnumbered by competing bacteria in raw foods. This competition may not occur, however, in heated foods, and unrestrained growth of staphylococci may ensue. A million staphylococci per milliliter or gram of perishable foods will be inactivated by 66 C maintained for at least 12 min or by 60 C at 78h to 83 min.

Heat resistance varies somewhat with the food and the strain of staphylococcus. *D* values at 60 C in custard are about 7.7 to 7.8 min, and in chicken a la king about 5.2 to 5.4 min. About 0.37 to 0.48 Mrad of gamma rays on moist foods will kill most of the staphylococci.

The sources from which the food-poisoning staphylococci enter foods are for the most part human or animal. The nasal passages of many persons are laden with these organisms, which are a common cause of sinus infections. Also, boils and infected wounds may be sources. The human skin apparently is a source of these bacteria

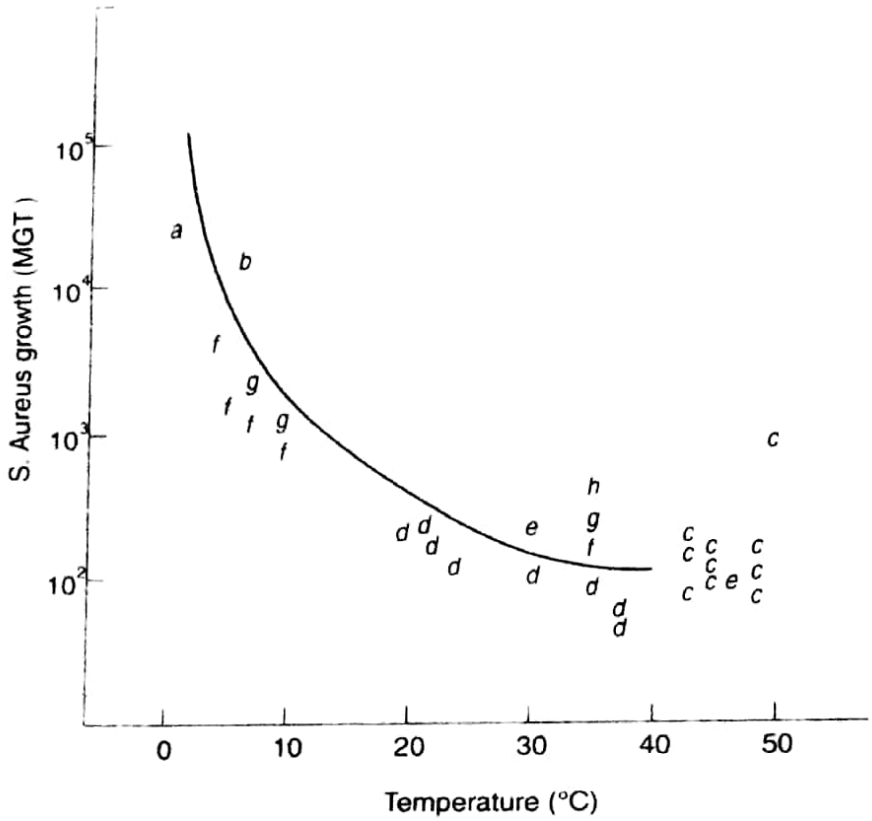

Fig. 24.5 *Mean generation times (MGT) of Staphylococcus aureus in (A) raw ground beef, (B) fresh raw eggs, (C) roast beef, (D) skim, whole, and raw milk, (E) barbequed chicken, (F) egg custard, (G) chicken a la king, and (H) ham salad. Regression coefficient, r = 0.89. (From Rivituso and Synder, 1980.)*

only when they have come from nasal passages or local infections. Staphylococci are becoming increasingly important in causing mastitis in cows, and some of these cocci can form enterotoxin in milk or milk products. Ordinarily, air is a relatively unimportant source of the cocci, except when they are being introduced there from human sources.

The Enterotoxin The staphylococcal enterotoxins are simple proteins with molecular weights between 26,000 and 30,000. The single polypeptide chains are cross-linked by a disulfide bridge to form a characteristic **cystine loop**. Since many of the amino acids in this loop are similar in each of the enterotoxin types, it is thought to be the toxic part of the molecule. Of the several types of enterotoxins, A and D are more often associated with food-poisoning outbreaks. Appreciable levels of enterotoxin are produced only after considerable growth of the staphylococcus; usually a population of at least several millions per milliliter or gram must be attained. Therefore, the conditions that favor toxin production are those best for growth of the staphylococcus. Toxin is produced at an appreciable rate at temperatures between 15.6 and 46.1 C, and production is best at 40 C. Under the best conditions enterotoxin may become evident within 4 to 6 hr. The lower the temperature during growth, the longer it will take to produce enough enterotoxin to cause poisoning. Enterotoxin has been demonstrated in a good culture medium in 3 days at 18 C and in 12 hr at 37 C but not in 3 days at 15 C, 7 days at 9 C, or 4 weeks at 4 to 6.7 C. Production of enterotoxin

by the staphylococci is more likely when competing microorganisms are absent, few, or inhibited for some reason. Therefore, a food that had been contaminated with the staphylococci after a heat process would be favorable for toxin production. There is evidence that toxin is produced by staphylococci growing in the intestinal tracts of patients when treatment with antibiotics has destroyed or inactivated other competitive bacteria there.

The type of food evidently has an influence on the amount of enterotoxin produced; little is produced in salmon, for example, and much in meat products and custard-filled bakery goods. The presence of starch and protein in considerable amounts is supposed to enhance toxin production by the staphylococci.

An important characteristic of the enterotoxins is their stability toward heat. Interpretation of data on heat inactivation of enterotoxin should include a consideration of the assay procedure employed. A discussion on the thermal stability of the enterotoxins is further complicated by (1) variation in the initial concentration of each type in foods, (2) the medium in which heated, and (3) the temperature of heating; some observations suggest greater thermal inactivation at lower temperatures than at higher temperatures. This has been explained by a low-temperature aggregation phenomenon. Additionally, low-temperature heat-inactivated enterotoxin can undergo reactivation in some foods. In general the heat treatments of pasteurization (72 C for 15 sec) and ultrahigh-temperature heating (143.3 C for 9 sec) would not be sufficient to inactivate the enterotoxins. Some of the characteristics of thermal inactivation are presented in Table 24.6. The effect of concentration and suspending medium is evident. Type B enterotoxin is the most heat-resistant, as indicated by the higher *z* value (Table 24.6). The normal cooking of foods will not destroy the toxin formed therein before the heat process. Such foods might cause poisoning, although no live staphylococci could be demonstrated.

Incidence of the Disease Reported cases in the United States are presented in Table 24.1. There probably are no reliable figures on the numbers of cases of staphylococcus poisoning in the United States or in any of the states for any given period. The poisoning usually is not reported or publicized unless the outbreak is fairly large, as at a picnic, large dinner, or convention. It is known, however, that a large percentage of all cases reported as food poisoning or food infection actually are staphylococcus poisoning and that many of us encounter this illness during our lifetime.

Foods Involved Of the many kinds of food that have been involved in causing staphylococcus food poisoning, custard-and cream-filled bakery goods, ham, and poultry have caused the most outbreaks. About 75 percent of all staphylococcal food-poisoning outbreaks occur because of inadequate cooling of foods. Other foods incriminated include other meats and meat products, fish and fish products, milk and milk products, cream sauces, salads, puddings, custards, pies, and salad dressings. The fillings in bakery goods usually are good culture media in which the staphylococci can grow during the time that these foods are held at room temperatures. Toxin production has even been reported in imitation cream filling. Tongue and mildly cured, rapidly cured, tenderized, or precooked hams, although perishable, are often held without adequate refrigeration, as had been done without difficulty with the old-style

Table 24.6 *Thermal Inactivation Characteristics of Staphylococcal Enterotoxins*

Type of enterotoxin	*Assay system used*	*Sensitivity or amount Injected or fed*	*Initial toxin concentration, µg/ml*	*Heating medium**	*Heating temp., F*	F_{250}, *min*	*z value, F*
A	Monkey feeding	47.25 µg	7	Distilled water	212–250	8	46
A	Cat; intravenous injection	2.3 µg	7	Distilled water	212–250	11	48
B	Serological; double diffusion tube	0.7 µg/ml	30	0.04 *M* veronal buffer, pH 7.2	204–259	19–16.4	55–58
	Cat; intravenous injection	2.0 µg	30	0.04 *M* veronal buffer, pH 7.2	240	32.5	
A	Serological; single diffusion tube	1 µg/ml	21	0.04 *M* veronal buffer, pH 7.2	212–250	22	50
B	Serological single diffusion tube	1 µg/ml	10–100	0.04 *M* veronal buffer, pH 7.2	250–320	62	52
					320	2.8	

*Dialyzed growth material dissolved in listed solvent.

Source: Summarized from various workers by Tatini (1976).

country-cured hams. If contaminated leftover turkey, or other fowl, along with the gravy and dressing, is kept out of the refrigerator, it may cause poisoning. Foods that ordinarily are too acid for good growth of the staphylococci may have this acidity reduced by added ingredients, such as eggs or cream, and then become dangerous. Growth and toxin production by staphylococci may take place in the steam tables in cafeterias and restaurants and in food-vending machines that keep foods heated for extended periods if temperatures and times are not properly controlled.

The Disease Individuals differ in their susceptibility to staphylococcus poisoning, so that of a group of people eating food containing toxin some may become very ill and a lucky few may be affected little or not at all. The incubation period (time between consumption of the food and appearance of the first symptoms) for this kind of poisoning usually is brief, 2 or 4 hr (ranging from 1 to 7 hr), differing in this respect from the other common food poisonings and infections, which usually have longer incubation periods.

The most common human symptoms are salivation, then nausea, vomiting, retching, abdominal cramping of varying severity, and diarrhea. Blood and mucus may be found in stools and vomitus in severe cases. Headache, muscular cramping, sweating, chills, prostration, weak pulse, shock, and shallow respiration may occur. Usually a subnormal body temperature is found rather than fever. The duration is brief, usually only a day or two, and recovery ordinarily is uneventful and complete. The mortality is extremely low. For the most part no treatment is given, except in extreme cases, when saline solutions may be given parenterally to restore the salt balance and counteract dehydration.

Outbreaks of food poisoning often are attributed to staphylococci on the basis of the type of food involved, the short incubation period, and perhaps the demonstration of the presence of stadphylococci in the food. An actual diagnosis of the poisoning would depend, however, on isolation of staphylococci and demonstration that these produce enterotoxin or isolation and detection of the enterotoxin.

Conditions Necessary for an Outbreak The following conditions are necessary for an outbreak of staphylococcus food poisoning: (1) The food must contain enterotoxin-producing staphylococci, (2) the food must be a good culture medium for growth and toxin production by the staphylococci, (3) the temperature must be favorable to growth of the cocci, and enough time must be allowed for production of enterotoxin, and (4) the enterotoxin-bearing food must be ingested.

Prevention of Outbreaks The means of prevention of outbreaks of staphylococcus food poisoning include (1) prevention of contamination of the food with the staphylococci, (2) prevention of the growth of the staphylococci, and (3) killing staphylococci in foods. Contamination of foods can be reduced by general methods of sanitation, by using ingredients free from the cocci, e.g., pasteurized rather than raw milk, and by keeping employees away from foods when these workers have staphylococcal infections in the form of colds, boils, carbuncles, etc. Growth of the cocci can be prevented by adequate refrigeration of foods and, in some instances, by adjustment to a more acid pH. Also the addition of a bacteriostatic substance, such as serine or an antibiotic, has been suggested. Some foods may be pasteurized to

kill the staphylococci before exposure of the foods to ordinary temperatures, e.g., pasteurization of custardfilled puffs and eclairs for 30 min at 190.6 to 218.3 C oven temperature.

❑ Salmonellosis

Salmonellosis may result following the ingestion of viable cells of a member of the genus *Salmonella*. It is the most frequently occurring bacterial food infection and in some years it is the most frequently occurring bacterial food-borne illness (Table 24.1). In addition to the typical food-poisoning salmonellosis syndrome, two other disease syndromes can result following consumption of salmonella, and they are compared in Table 24.7. Classification of the genus *Salmonella* is confusing, and the naming of organisms does not follow the usual rules of nomenclature. Historically, the names given to isolated salmonellae were related to their pathogenicity in people or animals–for example, *S. typhimurium*, responsible for typhoid in mice, and *S. typhi*, responsible for human typhoid. This approach gave way to naming based on the site or location of first isolation in human infections, for example, *S. london*, *S. panama*, and *S. stanleyville*. *Salmonella* isolates are currently identified using the Kauffman-White scheme, a serological procedure in which organisms can be represented by the numbers and letters of the different antigenic sites: O (somatic), Vi (capsular), and H (flagellar). This scheme only identifies antigens of diagnostic importance and does not provide a complete antigenic record of an isolate. The term **serovar** is used to distinguish strains of different antigenic complements. Further subdivision can be made to **biovars**, i.e., different sugar fermentation patterns shown by members of the same serovar. This detail is helpful in epidemiological investigations because serovars and biovars can be used as "markers" to trace the actual route of an outbreak to its source. There are currently over a thousand known serovars.

The *Salmonella* infections that are called food poisoning may be caused by any of a large number of serovars (Table 24.7). Usually the infecting bacterium has grown in the food to attain high numbers, increasing the likelihood of infection and often resulting in outbreaks in families or larger groups. By contrast, other intestinal pathogens, such as organisms causing the dysenteries and typhoid and paratyphoid fevers, usually have a longer incubation period before symptoms and, except under epidemic conditions, occur in only scattered cases.

The Organism The salmonellae are gram-negative non-spore-forming rods that ferment glucose, usually with gas, but usually do not ferment lactose or sucrose. Like other bacteria, they will grow over a wider range of temperature, pH, and a_w in a good culture medium rather than in a poor one. For example, minimal temperatures for growth in foods range from 6.7 to 7.8 C in chicken a la king to over 10 C in custard and ham salad. Their maximal temperature is about 45.6 C. They grow well at room temperatures, but their optimum is about 37 C. Mean generation times in several foods are presented in Figure 24.6. The pH range for growth is 4.1 to 9.0, growing in low-acid foods; salad at pH 5.5 to 5.7 has been found unfavorable for growth. The lowest a_w for growth varies with the food but is about 0.93 to 0.95. The species and strains of *Salmonella* differ, too, in heat resistance and in the effect of environmental

Table 24.7 *The Salmonellosis Syndromes in Humans*

Disease	*Etiologic agent*	*Nature of organism*	*Incubation period, signs, and symptoms*	*Source, reservoir, and epidemiology*
Salmonellosis	*Salmonella choleraesuis, enteritidis, typhimurium, heidelberg, derby, java, infantis, enteritidis, montevideo, etc.*	Has O (somatic) and two phases of H (flagellar) antigens, over 2,000 known serovars, but only about 50 occur commonly	5-72 hr, commonly 12–36 hr; diarrhea, abdominal pain, chills, fever, vomiting, dehydration, prostration, anorexia, headache, malaise; duration of several days; enteritis or local infection may also occur	Feces of infected domestic or wild animals and human beings; infants, aged, and malnourished persons and those with concomitant diseases are more susceptible; carrier state usually lasts a few days to a few weeks, but sometimes for months
Typhoid fever (enteric fever)	*Salmonella typhi*	Similar to other salmonellae but adapted to human host; has VI (capsular) an-tigens as well as O and H antigens	7–28 days, mean 14 days; in food out-breaks may be in shortest incubation range; septicemia and lymphoid tissue involvement; malaise, headache, high continued fever, cough, anorexia, nausea, vomiting, constipa-tion, slow pulse rate, tender and distended abdomen, enlarged spleen, nosebleed, rose spots on chest and trunk, perspiratton, chills, delirium, dulled sensorium, diarrhea, bleeding from bowel; relapses occur; slow convalescence of 1 to 8 weeks	Feces and urine of infected persons; carriers are important in transmission; some are long-term carriers; water also is involved in transmission
Paratyphoid fever (enteric fever)	*Salmonella enteritidis*, paratyphi A, paratyphi B, paratyphi C, *sendai*	Similar to other salmonellae but more or less adapted to human host	1–15 days; bloodstream infection; headache, continued fever, profuse perspiration, nausea, vomiting, abdominal pain, enlarged spleen, diarrhea, sometimes rose spots; milder and shorter duration (1–3 weeks) than typhoid	Feces and urine of infected persons; carriers important in transmission

Source: Adapted from: Bryan, F.L., 1982. Diseases tranamitted by foods. DHEW No. (CDC) 76-8237. Center for Disease Control, Atlanta, Ga.

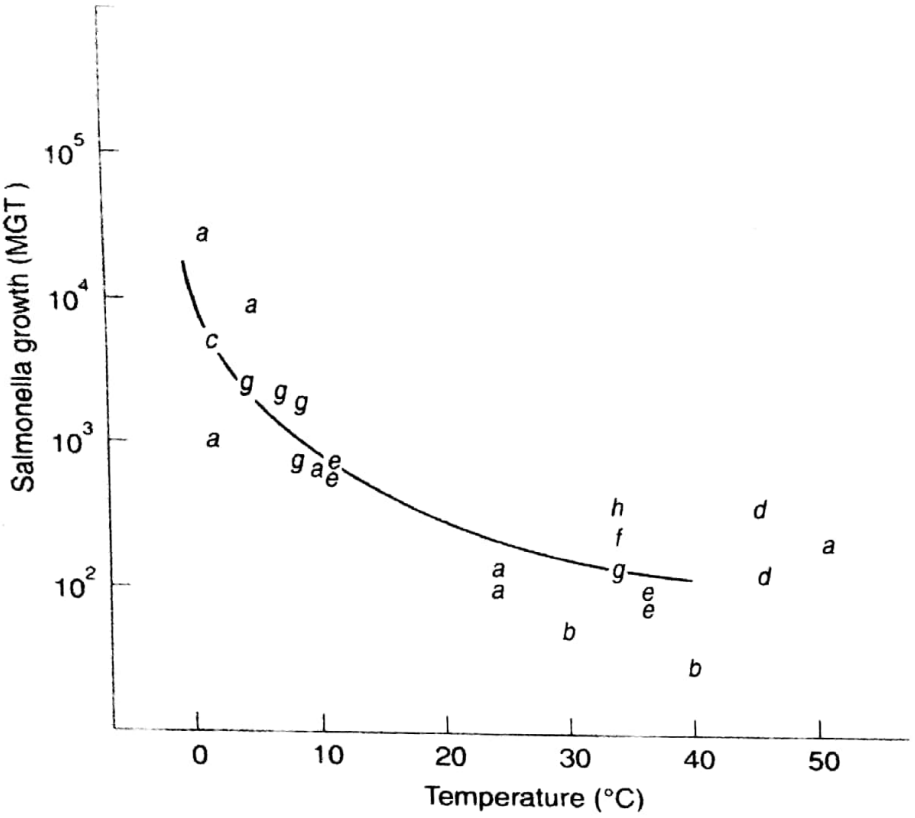

Fig. 24.6 *Mean generation times (MGT) of Salmonella in (A) raw ground beef, (B) barbequed chicken, (C) bacon, (D) roast baef, (E) milk, (F) egg custard, (G) chicken á la king, and (H) ham salad. Regression coefficient, r = 0.66. (From Rivituso and Synder, 1981.)*

factors on growth. Depending on the food and the serotype, the D_{60c} values range from 0.06 to 11.3 min. Recommendations for thermal destruction of salmonellae in perishable foods are similar to those for staphylococci, namely, heating to 66 C and holding all parts at that temperature for at least 12 min (or 78 to 83 min at 60 C). F_{140} values (minutes at 140 F necessary to reduce an inoculum to an undetectable level) found for two species were 78 and 19 min, respectively, in custard, and 81.5 and 3.1 min in chicken á la king. These results illustrate how the required heat treatment differs with the species of *Salmonella* and the food heated.

The likelihood of infection by consumption of a food containing salmonellae depends on the resistance of the consumer, the infectiveness of the particular strain of *Salmonella.* and the number of organisms ingested. Less infective species such as *S. pullorum* must be ingested in hundreds of millions or in billions to bring about infection, but considerably fewer (about a million) organisms of more infective species, e.g., *S. enteritidis,* usually would be sufficient. Salmonellae apparently can attain considerable numbers in foods without causing detectable alterations in appearance, odor, or even taste. Of course, the more of any of these pathogens the food contains, the greater the likelihood of infection on the person who eats the food and the shorter the incubation time.

Sources of Salmonella Human beings and animals are directly or indirectly the source of the contamination of foods with salmonellae. The organisms may come from actual cases of the disease or from carriers. Most frequently isolated serovars,

such as S. *typhimurium* and others (see Table 24.7) cause human gastroenteritis, but any of many other types may be responsible. The organisms also may come from cats, dogs, swine, and cattle, but more important sources for foods are poultry and their eggs and rodents. Chickens, turkeys, ducks, and geese may be infected with any of a large number of types of *Salmonella*, which are then found in the fecal matter, in eggs from the hens, and in the flesh of the dressed fowl. About one-third of all the food products involved in *Salmonella* outbreaks are meat and poultry products. Considerable attention has been given to shell eggs and to liquid, frozen, and dried eggs as sources of *Salmonella*. Infected rodents, rats and mice, may contaminate unprotected foods with their feces and thus spread *Salmonella* bacteria. Flies may play an important role in the spread of *Salmonella*, especially from contaminated fecal matter to foods. Roaches apparently also can spread the disease.

Changes in processing, packaging, and compounding of foods and feeds in recent years have resulted in an apparent increase in salmonellosis from these products. Salmonellae have been introduced by the incorporation of cracked and dried eggs in baked goods, candy, ice cream, and convenience foods such as cake and cookie mixes. The compounding of new food products may make possible the growth of salmonellae or other food-poisoning organisms, introduced by means of an ingredient in which they had been unable to grow, or these organisms may be in a product when sold and become able to grow in this food as it is modified for use. Large-scale handling of foods, as by commissaries or institutions, tends to increase the spread of trouble, and food vending machines add to the risk, as do precooked foods.

Feeds, especially those from meat or fish by-products, may carry salmonellae to poultry or meat animals. Even pet feeds have been known to transmit salmonellae to domestic animals, from which children have been infected.

Incidence of the Disease Numbers of reported human cases of salmonellosis (other than typhoid and paratyphoid fever) in the United States during recent years are given in Table 24.1 and Figure 24.7. Like most food-poisoning syndromes, the reported number of cases probably represents only a small percentage of the number of cases actually occurring.

Food Involved A large variety of foods are involved in causing outbreaks of *Salmonella* infections. Most commonly incriminated are various kinds of meats, poultry and products from them, especially if they are held unrefrigerated for long periods. Fresh meats may carry *Salmonella* bacteria that caused disease in the slaughtered animals or may be contaminated by handlers. Meat products, such as meat pies, hash, sausages, cured meats (ham, bacon, and tongue), sandwiches, and chili, often are allowed to stand at room temperatures, permitting the growth of salmonellae. Poultry and its dressing and gravy should not give trouble If properly handled and cooked but often are mishandled, as are fish and other seafood and products from them. Milk and milk products, including fresh milk, fermented milks, ice cream, and cheese, have caused infections. Since eggs may carry the salmonellae, foods made with eggs and not sufficiently cooked or pasteurized may carry live organisms, e.g., pastries filled with cream or custard, cream cakes, baked Alaska, and eggnog.

The Disease As with other infectious diseases, individuals differ in their susceptibility to *Salmonella* infections, but in general morbidity is high in any outbreak. As

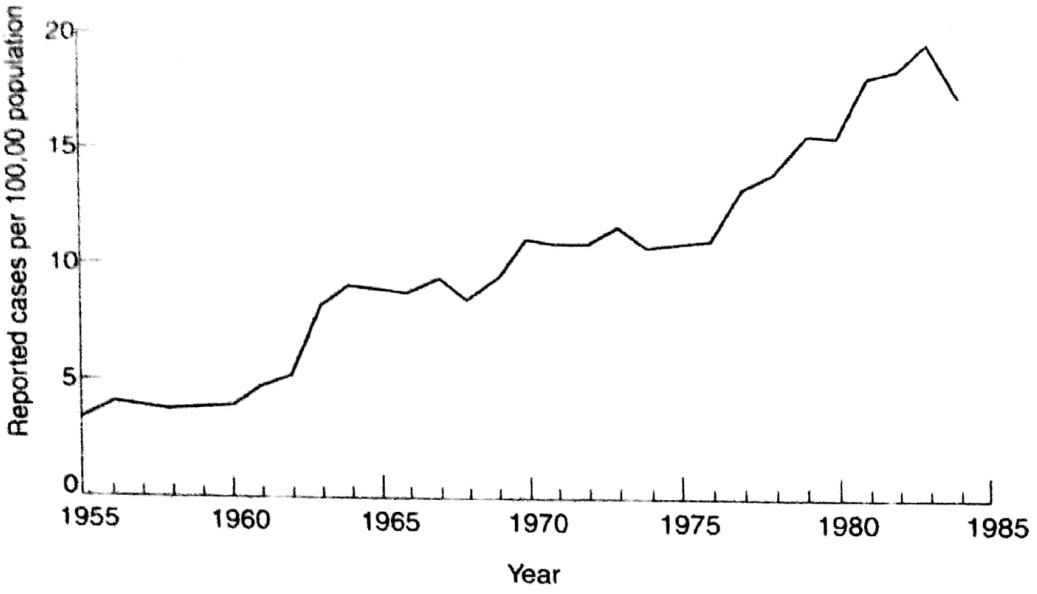

Fig. 24.7 *Reported cases of salmonellosis by year in the United States, 1955–1984. A slight decrease in reported cases of human salmonellosis was noted in 1984. This decrease most likely represents annual variation rather than a reversal of the secular trend toward increasing rates of salmonellosis in the United States. This steady increase in reported rates is thought to reflect increasing incidence of the disease rather than more efficient reporting. (Centers for Disease Control, Annual Summaries for 1984, 1986.)*

has been stated, the susceptibility of humans varies with the species and strain of the organism and the total numbers of bacteria ingested.

A longer incubation period usually distinguishes salmonellosis from staphylococcus poisoning: usually 12 to 36 hr for the former and about 2 to 4 hr for the latter. Shorter (as little as 5 hr) or longer (up to 72 hr) incubation periods may occur in some cases of *Salmonella* infections.

The principal symptoms of a *Salmonella* gastrointestinal infection are nausea, vomiting, abdominal pain, and diarrhea that usually appear suddenly. This may be preceded by a headache and chills. Other evidences of the disease are watery, greenish foul-smelling stools, prostration, muscular weakness, faintness, usually a moderate fever, restlessness, twitching, and drowsiness. The mortality is low, being less than I percent. The severity and duration vary not only with the amount of food eaten and hence the numbers of *Salmonella* bacteria ingested but also with the individual. Intensity may vary from slight discomfort and diarrhea to death in 2 to 6 days. Usually the symptoms persist for 2 to 3 days, followed by uncomplicated recovery, but they may linger for weeks or months. About 0.2 to 5 percent of the patients may become carriers of the *Salmonella* organism.

The laboratory diagnosis of the disease is difficult unless *Salmonella* can be isolated from the suspected food and from the stools of individuals. Often, however, the incriminated foods are no longer available, and the organisms disappear from the intestinal tract.

Conditions Necessary for an Outbreak The following conditions are necessary for an outbreak of a food-borne *Salmonella* gastrointestinal infection: (1) The food must contain or become contaminated with the *Salmonella* bacteria, (2) these

bacteria must be there in considerable numbers, either because of contamination or more often because of growth; these high numbers mean that the food must be a good culture medium, the temperature must be favorable, and enough time must be allowed for appreciable growth, and (3) the viable organisms must be ingested.

Prevention of Outbreaks Three main principles are involved in the prevention of outbreaks of food-borne *Salmonella* infections: (1) avoidance of contamination of the food with salmonellae from sources such as diseased human beings and animals and carriers and ingredients carrying the organisms, e.g., contaminated eggs, (2) destruction of the organisms in foods by heat (or other means) when possible, as by cooking or pasteurization, paying special attention to held-over foods, and (3) prevention of the growth of *Salmonella* in foods by adequate refrigeration or by other means. In the prevention of contamination, care and cleanliness in food handling and preparation are important. The food handlers should be healthy (and not be carriers) and clean. Rats and other vermin and insects should be kept away from the food. Ingredients used in foods should be free of salmonellae, if possible. Of course foods should not be allowed to stand at room temperature for any length of time, but if this happens, thorough cooking will destroy the *Salmonella* organisms (but not staphylococcus enterotoxin). Warmed-over leftovers, held without refrigeration, often support the growth of *Salmonella*, as may canned foods that have been contaminated and held after the cans were opened. Inspection of animals and meats at packing houses may remove some *Salmonella-infected* meats but is not in itself a successful method for the prevention of human salmonellosis.

❑ Clostridium perfringens Gastroenteritis

First reported in the United States in 1945, *Clostridium perfringens* gastroenteritis is being detected and reported more frequently than formerly, although it probably is no more prevalent (Table 24.1).

The Organism The bacterium causing this Illness is *C. perfringens (welchii)*, type A, a gram-positive, nonmotile, anaerobic, spore-forming rod. Maximal temperature for growth is about 55 C, and optimal temperature is about 43 to 47 C. Growth is restricted at 15 to 20 C. Mean generation times in several foods are illustrated in Figure 24.8. The organism will not grow below pH 5.0 or above pH 9.0. It is inhibited by 5 percent NaCl ($a_w = 0.97$), *and some strains are held back by* 2.5 *percent sodium nitrate.*

The spores of food-poisoning strains differ considerably in their heat resistance: $D_{90C} = 0.015$ to 8.71 min.

Foods Involved The spores have been found in part of the samples of most raw foods examined as well as in soil, sewage, and animal feces. Most commonly involved are meats that have been cooked, allowed to cool slowly, and then held for some time before consumption. Meat and poultry products account for about three-quarters of outbreaks attributed to *C. perfringens*. Fish paste and cold chicken also have been incriminated.

Since the spores are fairly common in raw foods and are heat-resistant, their presence in many foods may be unavoidable. Cooking of foods will destroy the

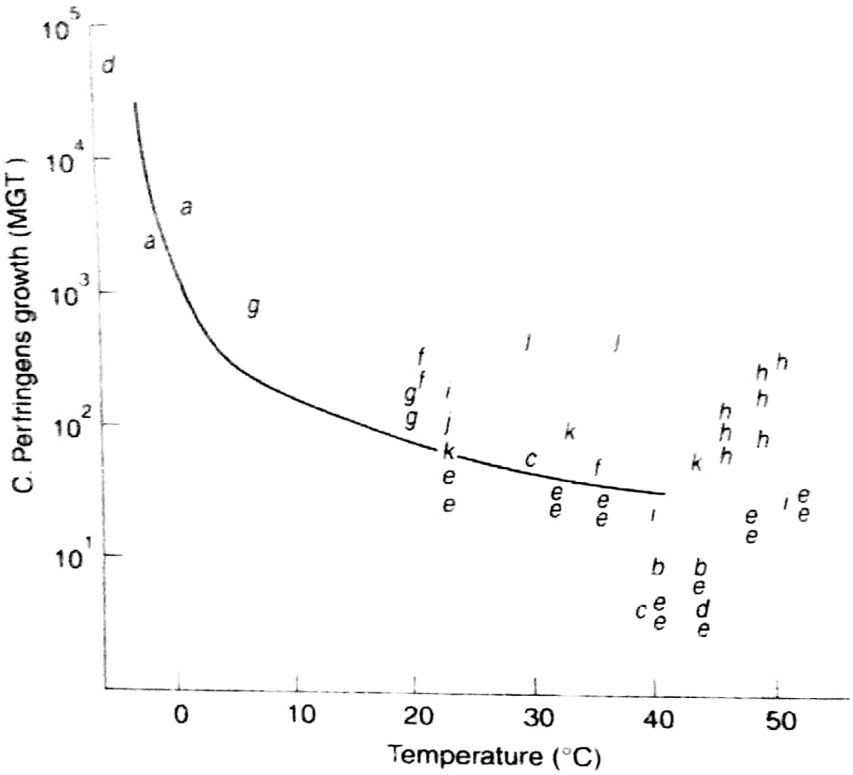

Fig. 24.8 *Mean generation times (MGT) of Clostridium perfringenes in (A) fresh chicken, (B) rolled beef strips, (C) barbequed chicken, (D) raw ground beef, (E) cooked ground beef, (F) turkey rolls, (G) ground beef casseroles, (H) roast beef, (I) cooked meat, (J) turkey rice soup, and (K) mashed potatoes. Regression coefficient, r = 0.71. (From Rivituso and Synder, 1981)*

vegetative cells and the spores of some strains; however, germination and out growth of surviving spores are possible in cooked foods which have been in adequately refrigerated. A typical outbreak might involve a large cut of roasted meat. The oven cooking would not kill all the spores but might create favorable reduced conditions. Following cooking and in the absence of proper cooling, the spores of *C. perfringens* germinate and their numbers increase. The presence of a large number of *C. perfringens* in a cooked food is indicative of mishandling.

The Disease The symptoms, which appear usually in 8 to 24 hr (median 12 hr) following ingestion include acute abdominal pain, diarrhea, and gas; fever, nausea, and vomiting are rare. Most outbreaks suggest that the ingestion of millions of viable cells of *C. perfringens* per gram of food are required for symptoms to occur. A toxin (enterotoxin) is released in the gut during sporulation of the cells and results in excessive fluid accumulation in the intestinal lumen. The enterotoxin is relatively heat-sensitive, being inactivated at 60 C for 10 min.

Conditions Necessary for an Outbreak The following conditions are necessary for an outbreak of *C. perfringens* gastrointestinal infection: (1) The food contains or becomes contaminated with *C. perfringens*, (2) usually the food is cooked and reduced conditions develop, (3) the food is inadequately cooled, and favorable temperatures and enough time are allowed for appreciable growth, (4) the food is consumed without reheating so that large numbers of viable cells are ingested, and (5) the cells sporulate in vivo and elaborate the enterotoxin.

Prevention of Outbreaks Means of prevention of outbreaks of *C. perfringens* food infection include (1) adequate and rapid cooling of cooked meats and other foods, (2) holding hot foods above 60 C, (3) reheating of leftover foods, and (4) good personal hygiene.

❑ *Vibrio Parahaemolyticus* Infection and Other Vibrios

Outbreaks of *V. parahaemolyticus* gastroenteritis have received considerable attention in Japan, where it is one of the most commonly occurring food-poisoning syndromes. In recent years, this organism has been responsible for several food-borne outbreaks in the United States (Table 24.1).

V. parahaemolyticus is a gram-negative straight or curved motile rod. It is a halophile (requiring 1 to 3 percent NaCl) and will grow in 7.0 percent NaCl. Optimal temperature for growth is 35 to 37 C, but it will grow over a range of 10 to 44 C. Growth is inhibited below pH 5.0 and above pH 11.0. *V. parahaemolyticus* has been isolated from many seafoods, including oysters, shrimp, and blue crabs. The organism can readily be killed by proper cooking of the seafood. Most outbreaks in this country occur because raw or uncooked seafood is allowed to contaminate and reinoculate cooked seafood. Holding recontaminated cooked seafood at too high temperatures permits rapid growth and a subsequent high population of *V. parahaemolyticus*. If the food is not reheated, signs and symptoms will result. Some of the characteristics of *V. para Vibrio cholerae* serovar O group 1 is responsible for epidemic or Asiatic cholera. Cholera is an extremely serious worldwide, usually water-borne disease. A limited number of outbreaks in the continental United States have been identified as being transmitted by seafoods. Other serovars, not O1, do not represent the same health threat as does *Vibrio cholerae* O1 but can be responsible for intestional infection or gastroenteritis. These *Vibrio cholerae* strains, which closely resemble O1, are referred to as nonagglutinable (NAG) vibrios because they do not react or agglutinate in the anti-O1 serum. Several surveys conducted in the Chesapeake Bay and along the gulf states suggest that both O1 and and non-O1 *Vibrio cholerae* are fairly common in estuary water. Food-borne outbreaks of *Vibrio cholerae* and non-O1 strains have been associated with the consumption of oysters.

Vibrio vulnificus can also be isolated from seafoods and seawater. The organism is highly invasive, releasing both a hemolysin and a cytotoxin, and can result in primary septicemia in humans.

❑ Enteropathogenic *Escherichia Coli*

E. coli is generally regarded as part of the normal flora of the human intestinal tract and that of many animals. Several nursery epidemics in the 1940s implicated *E. coli* in diarrheal disease in infants. Serotypes of *E. coli* which have been implicated in human diarrheal diseases or food-poisoning outbreaks have been designated enteropathogenic *E. coli* (EEC). The human disease syndromes resulting from the ingestion of EEC have been divided into two main groups. The first group consists of strains which produce an enterotoxin and result in a choleralike or enterotoxigenic illness in humans (see Table 24.8). These enterotoxigenic strains usually produce two enterotoxins, a heat-stable (ST) and a heat-labile (LT) toxin, and are thought to

Table 24.8 *Characteristics of some Food-borne Illnesses*

Disease	Etiologic agent	Incubation period, signs, and symptoms	Foods Involved	Control measures
Vibrio parahaemolyticus infection	*V. parahaemolyticus*	2-48 hr, usually 12 hr; abdominal pain, diarrhea (watery stools containing blood and mucus), usually nausea and vomiting, mild fever, chills, headache, prostration; recovery within 2–5 days	Raw foods of marine origin; saltwater fish, shellfish, crustacea, and fish products	Cook foods thoroughly; chill foods rapidly in small quantities; prevent cross contamination from saltwater fish; sanitize equipment; avoid using seawater for rinsing foods to be eaten raw or for cleaning
Enteropathogenic *Escherichia coli* infection	E. *coli;* both enterotoxigenic and invasive strains cause illness	8–24 hr, mean 11 hr (invasive type); 8–44 hr, mean 26 hr (enterotoxigenic type); invasive illness: fever, chills, headache, myalgia, abdominal cramps, profuse, watery diarrhea; similar to shigellosis; enterotoxigenic illness: diarrhea (rice-water stools), vomiting, dehydration, shock; similar to cholera	Coffee substitute, salmon(?), cheese	Chill foods rapidly in small quantities; cook foods thoroughly; practice personal hygiene; prepare foods in sanitary manner; protect and treat water; dispose of sewage in sanitary manner
Bacillus cereus gastroenteritis	Diarrheal, syndrome, diarrheagenic toxin	8–16 hr or 1.5–5 hr; nausea, abdominal cramps, watery diarrhea, some vomiting	Custards, cereal products, puddings; sauces, meat loaf	Chill foods rapidly in small quantities; hold hot foods at 65 C or above; practice personal hygiene; process and prepare food in sanitary manner; reheat leftover foods to 71.1 C
	Emetic syndrome, emetic toxin	Incubation period 1–6 hr, 2–5 most common, with nausea and vomiting predominant, similar to staphylococcal intoxication); short duration, 1 day or less	Fried rice, mashed potatoes, vegetable sprouts	

Contd

Contd.

Shigellosis (bacillary dysentery)	*Shigella sonnei*, S. *flexneri*, S. *dysenteriae*, S. *boydii*	1–7 days, usually less than 4 days; extremely variable, mild to severe symptoms: abdominal cramps, fever, chills, diarrhea, watery stools (frequently containing blood, mucus, or pus), tenesmus, headache, lassitude, prostration, nausea, de-hydration	Moist, mixed foods; milk, beans; potato, tuna, shrimp, turkey, and macaroni salads; apple cider; and poi	Practice personal hygiene; chill foods rapidly in small quantities; prepare food in sanitary manner; cook foods thoroughly; protect and treat water; dispose of sewage in sanitary manner; control flies
Yersiniosis	*Yersinia pseudotuberculosis, Y enterocolitica*	24–36 hr and longer; abdominal pain suggesting acute appendicitis, fever, headache, malaise, anorexia, diarrhea, vomiting, nausea, chills, pharyngitis, leukocytosis, erythema nodosum	Pork and other meats, raw milk, or any contaminated raw or leftover food	Cook foods thoroughly; protect foods from contamination; control rodents
Arizona infection	*Arizona hinshawii*	2–46 hr, usually 24 hr; abdominal pain, diarrhea, nausea, chills, headache, weakness, fever, lasts a few days	Turkey, chicken, cream-filled pastry, ice cream, custard containing eggs	Chill foods rapidly in small quanti- ties; cook foods thoroughly; prevent fecal contamination; avoid cross contamination from raw to cooked foods; sanitize equipment; reheat leftover foods thoroughly
Beta-hemolytic streptococcal infections (scarlet fever, septic sore throat)	*Streptococcus pyogenes*	1–3 days; sore and red throat, pain in swallowing, tonsillitis, high fever, headache, nausea, vomiting, malaise, rhinorrhea; occasionally a rash occurs	Milk, ice cream, eggs, steamed lobster, potato salad, egg salad, custard, and pudding; foods usually contain eggs or milk	Chill foods rapidly in small quanti ties; practice personal hygiene; cook foods thoroughly; pasteurize milk, exclude workers with respiratory illness or skin lesions

Source: Adapted from Bryan, F.L. 1982. Diseases transmitted by foods. DHEW No. (CDC) 76-8237. Center for Disease Control, Atlanta, Ga.

be responsible for infantile diarrheal diseases and traveler's diarrhea. To experience the enterotoxigenic illnesses, EEC serotypes capable of elaborating the enterotoxins must be ingested, followed by colonization in the upper small intestine and production of the enterotoxins. The enterotoxins apparently mediate a net movement of water into the intestinal lumen. This fluid accumulation occurs without any gross macroscopic change in the intestinal epithelium and in the absence of penetration or invasion by the bacteria.

The second major group consists of invasive strains which produce a cytotoxin and result in the invasive illness, colitis, or dysenterylike syndrome. These serotypes are nonenterotoxigenic, grow in the colon, and invade or penetrate the epithelial cells of colonic mucosa, resulting in the signs and symptoms outlined in Table 24.8.

A large infective dose of EEC is required for either the enterotoxigenic or invasive illness to occur. Therefore, foods must be highly contaminated or inadequately preserved or refrigerated to allow for prolific growth. The optimal temperature for growth is 37 C, with a temperature range for growth of 10 to 40 C. The optimal pH for growth is 7.0 to 7.5, with the minimum at pH 4.0 and the maximum at pH 8.5. The organism is relatively heat sensitive and can readily be destroyed at pasteurization temperatures and by the proper cooking of foods. Table 24.8 lists some of the foods implicated in EEC outbreaks and some methods of prevention. In addition to the above strains, there is a group referred to as hemorrhagic *E. coli*. These strains can result in illness in humans as manifested by bloody diarrhea and severe abdominal pain.

❑ Bacillus Cereus

Reports of outbreaks of *B. cereus* food gastroenteritis are quite uncommon in the United States; however, many European countries report frequent implication of this organism in food-borne illness. *B. cereus* is a gram-positive, aerobic, spore-forming rod. Its optimal temperature for growth is 30 C, with a minimal temperature for growth at 10 C and a maximum of 49 C. The pH range for growth is 4.9 to 9.3. *B. cereus* spores have reported *D* values (heat resistance) of D_{100C} of 2.7 to 3.1 min in skim milk and D_{100C} of 8 min in phosphate buffer (pH 7.0). Numerous surveys on foods and ingredients have indicated a high percentage of samples containing *B. cereus*. It is undoubtedly widely distributed in nature and our food supply.

Extremely large numbers (10^8 per gram) of viable cells of *B. cereus* must be ingested to develop signs and symptoms (Table 24.8) of the syndrome. Two syndromes are recognized: the diarrheal syndrome and the emetic syndrome. Some of the characteristics of *B. cereus* gastroenteritis are presented in Table 24.8.

❑ Shigellosis

Food-borne outbreaks of shigellosis have been reported in the United States, but most incidents of shigellosis involve contaminated water.

Optimal temperature for growth is 37 C, with a temperature range of 10 to 40 C. The organisms tolerate salt concentrations of 5 to 6 percent and are relatively heat sensitive. Pathogenicity involves the release of a lipopolysaccharide endotoxin which affects the intestinal mucosa. Some of the characteristics of this food-borne illness are presented in Table 24.8, and numbers of cases are shown in Figure 24.9.

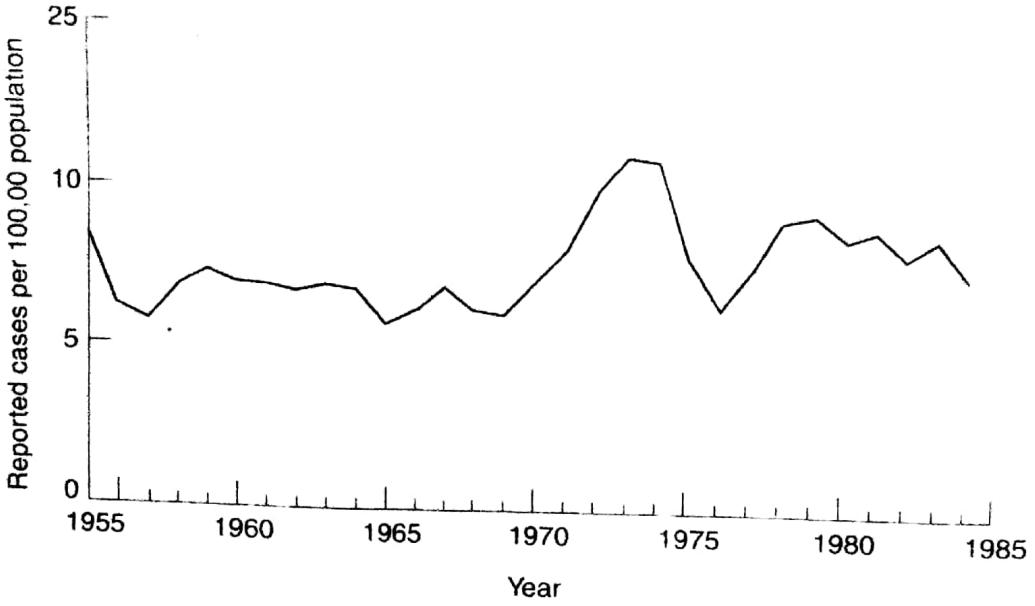

Fig. 24.9 *Reported cases of shigellosis by year in the United States, 1955–1984. For 1984, 17, 371 cases of shigellosis were reported in the United States. Approximately 70 percent of the Shigella isolates reported to the CDC each year are Shigella sonnei, with Shigella fexneri accounting for a large percentage of the rest. (Centers for Disease Control, Annual Summaries for 1984, 1986.)*

❑ Yersinia Enterocolitica

Yersinia enterocolitica is a small rod-shaped bacteria that can cause gastrointestinal illness in humans. It has been isolated from the intestinal tracts and feces of many animals, including cats, pigs, dogs, deer, raccoons, and horses. The pig appears to be the main reservoir for the strains causing infection in humans. *Y. enterocolitica* has been isolated from many foods, including beef, pork, liquid eggs, soft cheese, raw milk, pasteurized milk, fish, raw oysters, shrimps, crabs, chocolate milk, turkey, chow mein, powdered milk, and tofu.

Some strains are known as environmental or noninvasive types and are not known to cause illness in humans. The invasive or virulent strains of *Y. enterocolitica* are responsible for gastroenteritis in humans.

The usual symptoms, including severe abdominal pain, fever, and diarrhea, occur 24 to 36 hr after consumption of the product. Occasionally, the incubation time may be considerably longer. The abdominal discomfort is quite specific and usually manifests itself as a sharp pain in the lower right quadrant of the abdomen. For this reason it has frequently been described as pseudoappendicitis. In one outbreak sixteen children had appendectomies.

Although the organism has been isolated from many foods, there have been relatively few food-borne outbreaks attributed to *Y. enterocolitica*. The isolation from pasteurized milk is probably the result of postpasteurization contamination, since even the most heat-resistant strains are reported to be killed by pasteurization. The unique characteristic of the organism is its ability to grow at commercial refrigeration temperatures, i.e., less than 5 C.

A closely related organism, *Y. pseudotuberculosis*, has also been implicated as an etiological agent in human illness. Both *Y. enterocolitica* and *Y. pseudotuberculosis* are currently classified as members of the family Enterobacteriaceae.

❑ Campylobacter

The heat-tolerant *Campylobacter*, i.e., *C. jejuni* and *C. coli*, are the strains most frequently associated with acute gastroenteritis in humans. The term "heat- tolerant" is a misnomer since it refers to growth at 42 C and not to any degree of heat tolerance or heat resistance; actually, these strains are inactivated at temperatures above 45 to 50 C. Their most distinguishing characteristics are absence of growth below 25 C, sensitivity to air (21 percent O_2), resistance to cephalothin, a generation or doubling time of about 1 hr at optimum temperature (42 C), sensitivity to drying, sensitivity to acid conditions, and being a poor competitor in mixed populations because of an inability to utilize carbohydrates. It is therefore very unlikely that they can grow to large populations in foods held at or below room temperature.

C. jejuni is associated with warm-blooded animals, and many animals and animal by-products used for human and animal feeds are contaminated with the organism. Various strains have been isolated from chicken carcasses and feces, swine carcasses and feces, sheep carcasses and feces, turkeys, pork sausages, various red meats, and ground beef.

Many researchers have suggested that *C. jejuni* and *C. coli* are a major cause of a large number of cases of bacterial enteritis worldwide. Limited specific outbreaks have been reported; the products incriminated include raw milk, raw clams, undercooked chicken, contaminated pasteurized milk, cake icing, raw hamburger, and barbecued chicken. However, campylobacters are isolated from the feces of patients experiencing gastroenteritis at a greater rate than are salmonellae. For this reason it is suspected that the actual number of cases is much higher than the number reported.

Little is known about the actual mechanism of pathogenicity in humans; however, at least some strains produce a heat-labile enterotoxin. The organism is also invasive. Furthermore, it is thought that the organism is highly virulent since the actual numbers in foods are quite low.

Symptoms include abdominal pain, cramps, diarrhea, headache, fever, and occasionally bloody stools. The incubation time is usually 2 to 3 days but may be as long as 7 to 10 days.

Pasteurization of milk or the proper cooking of foods prior to consumption would eliminate many outbreaks. Since *Campylobacter* are widespread in foods of animal origin care must be taken to avoid cross contamination from raw meats to cooked items.

❑ Plesiomonas Shigelloides

Plesiomonas shigelloides is a facultatively anaerobic gram-negative rod. Most strains are motile by polar flagella and can grow on minimal media containing ammonium salts as the nitrogen source and glucose as the carbon source. The organism can be pathogenic in humans, causing disturbances of the intestinal tract. Diarrhea in

various degrees of severity is the major symptom. The organism has been isolated from the aquatic environment, fish, oysters, and crabs and from mammals such as cats, dogs, zoo animals, goats, sheep, and monkeys.

P. shigelloides has been implicated in several food-borne outbreaks involving oysters, crabs, and fish. The organism can be isolated from the feces of individuals with no apparent symptoms. However, it is thought that *P shigelloides* is an opportunistic pathogen and not a normal member of the human intestinal flora. Miller and Koburger (1986) examined forty environmental isolates of *P. shigelloides* and noted that all strains grew at pH 4.5 and 8.5. Fifty-eight percent of the isolates grew at pH 4.0, 22 percent grew at 8 C, and 25 percent grew at 45 C. All the isolates were inactivated by heating at 60 C for 30 min. Additionally, all the isolates grew in 3% sodium chloride, and some strains even grew in 5% sodium chloride. All the isolates were inactivated by heating at 60 C for 30 min.

❑ Listeria Monocytogenes

Listeria monocytogens is a gram-positive, motile, short rod capable of growth at 4 C. In cattle, it can result in abortion and mastitis, and the infected animals will shed the organism in the milk. Other infected animals, including sheep and chickens, can serve as a source of the organism in the food supply. *L. monocytogenes* has been isolated from water, milk, silage, sewage, and the feces of many animals, including humans.

The first cases of human listeriosis were reported in 1929. Human infections usually manifest themselves as an infection in a pregnant woman, a fetus, or a newborn infant. In adults, the disease occurs most frequently in immunecompromised individuals and is characterized as septicemia, meningitis, or men-ingoencephalitis.

Major food-borne outbreaks have been reported in various countries. Table 24.9 outlines three outbreaks in the United States. The contaminated coleslaw had been made from cabbage which had been fertilized with sheep manure. Several of the sheep had experienced ovine listerosis. Following harvesting, the cabbage was held in cold storage; this prolonged low-temperature condition probably enriched for *L. monocytogenes.*

Table 24.9 *Three Outbreaks of Listeria Monocytogenes in the United States*

Date	Location	Food vehicle	Cases	Fatality rate, %
1981	Nova Scotia	Coleslaw	34 P* 7A	44
1983	Massachusetts	Milk†	7 P 42 A‡	29
1985	Los Angeles County	Soft cheese§	93 P 49 A	33

* A = adult; P = perinatal.

† Pasteurized.

‡ Most were immunocompromised.

§ Mexican-style.

Like many gram-positive organisms, it is capable of persisting in the soil, and the use of manure containing listeria may represent a significant concern when applied to cropland, particularly when the products are fruits or vegetables which may not undergo additional heat treatments.

The presence of *L. monocytogenes* in pasteurized milk raised considerable concern about the adequacy of milk pasteurization. At least two reports (Bradshaw et al., 1985; Donnelly and Briggs, 1986) have suggested that proper pasteurization of milk will eliminate the organism. It is proposed that the contamination of milk is a postpasteurization problem. In 1986 the FDA concentrated on surveillance for the organism in the dairy processing plant environment and in finished products. *L. monocytogenes* and *L. inocula* were obtained from many environmental sources and from some finished products. At this time, no major studies on the thermal resistance of listeria in naturally infected cows have been published, although there has been an indication that the organism may survive the minimum times and temperatures established for milk HTST pasteurization, i.e., 161 F for 15 sec. The psychrotrophic growth characteristics of this organism suggest that contamination of a refrigerated food could represent a significant health hazard.

❑ Aeromonas Hydrophilia

A. hydrophilia is a gram-negative motile rod with an optimum temperature of 28 C. It may be pathogenic for frogs, fish, and mammals including humans (Krieg, 1984). The organism has rarely been incriminated in food-borne outbreaks in the United States; however, it is a newly recognized pathogen that may cause food-borne diarrhea. It is ubiquitous in water and has been reported to be the cause of outbreaks in other countries.

❑ Other Bacterial Food-borne Infections

Streptococcus faecalis and closely related species have been blamed for food illnesses, and since no enterotoxin could be demonstrated, the organisms were presumed but not proved to cause an infection. Foods reported to be involved in a limited number of outbreaks included barbecued beef, beef croquettes, Vienna sausage, ham bologna, turkey dressing, turkey a la king, Albanian and other cheeses, charlotte russe, and evaporated milk. Enterococci were isolated from most of these foods. The incubation period has been given as from 2 to 18 hr, and the symptoms as nausea, vomiting, and diarrhea. Formerly it was thought that only certain strains caused poisoning, but most authorities now maintain that all the accused organisms are innocuous. Volunteers have been fed massive doses of the organisms, including some supposedly incriminated in food-poisoning outbreaks, and also their products without any resulting enteritis.

For other organisms proof of implication in food-borne outbreaks is more substantial, e.g., *Arizona hinshawii* and *Streptococcus pyogenes*. The disease syndromes resulting from the ingestion of these bacteria are summarized in Table 24.8.

The food infections just discussed have been those involving bacteria able to grow in foods and hence to increase the dosage of the pathogen delivered to the person eating the food. Pathogenic organisms unable to grow in the food as it ordinarily

is handled may be carried by it, the food in this instance serving merely as an inactive carrier of the disease organisms, much as a doorknob, handkerchief, bus strap, drinking cup, or other forms would serve that purpose. Most of the diseases so transmitted are intestinal or respiratory. Some of the intestinal diseases so transported are typhoid and paratyphoid fevers, bacillary and amebic dysenteries, and cholera. Disease organisms from the throat and respiratory tract are those causing tuberculosis, scarlet fever, diphtheria, and other diseases. Also reportedly spread by foods are brucellosis, tularemia, Q fever, scarlet fever, septic sore throat, and infectious hepatitis.

Foods may be contaminated with disease organisms from food handlers, food utensils (eating, drinking, and kitchen), air, soil, water (e.g., oysters), the animal from which meat or milk came, and vermin such as flies, roaches, and rodents. Especially likely to be important are the people who handle foods after pasteurization, cooking, or other processing, e.g., cooks and helpers in the kitchen; waiters in the eating place; preparers and salespersons of unwrapped foods such as bulk candies, ice cream sandwiches and cones, baked goods, hot dogs, and hamburgers; and vendors of frozen desserts from counter freezers or other bulk sources.

Foods consumed raw, of course, are possible sources of pathogens. Thus, fresh fruits or vegetables may carry pathogens from a diseased handler to a healthy consumer, although few instances of such a transfer have been proved. Where night soil is used as fertilizer, there is a great risk of the presence of intestinal pathogens on the surface of fresh salad greens. Depending on the heat treatment administered, the cooking of foods may or may not destroy all pathogens present. Usually all on the surface will be killed, but not always those in the interior. Instances have been reported where even comparatively non-heat-resistant pathogens have survived cooking and caused an outbreak of disease.

The principle to be applied in the prevention of food-borne infections is to prevent the transfer of the pathogen from its source to the food, preferably by elimination of that source, but this objective is not always readily attained. Contamination from vermin can be prevented by their eradication; use of night soil for fertilizing soil for growing plant foods to be consumed raw can be prohibited; fruits or vegetables to be eaten raw can be washed thoroughly with water or with chlorine solution; shellfish from polluted waters can be rejected; and milk from diseased cows can be refused. However, meat animals or fod handlers that are diseased or are carriers are not always easy to detect. Methods for the detection of disease or disease organisms are for the most part laborious, difficult, and not always reliable; usually the tests are impractical to apply to meat animals at the packing plant and cannot be applied to food handlers as often as desirable. Of course, food handlers should not be permitted to work while they are ill or recovering, but they may be only mildly ill or be carriers and therefore may be allowed to work while giving off pathogens.

Many municipalities have ordinances which state that no person who is affected with any disease in a communicable form or is a carrier of such disease shall work in any eating or drinking establishment or be hired there, but the difficulty lies in enforcement of the ordinances. In most plants where foods are handled and processed, however, any check on the health of the workers is at present primarily the responsibility of the employer.